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original research

Resting Arterial Diameter and Blood Flow Changes With Resistance Training and Detraining in Healthy Young Individuals

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Context: Disruptions to habitual training routines are commonly due to injury or illness and can often lead to detraining adaptations. The implications of such adaptations to the human vasculature in a trained, asymptomatic population are not fully understood.

Objective: To determine the extent of local and systemic changes in arterial diameter and blood flow to resistance training and subsequent detraining in young adults.

Design: Randomized controlled clinical trial.

Setting: University physiology laboratory and fitness suite.

Patients or Other Participants: Twenty-one healthy volunteers (aged 20.0 ± 2.8 years, 11 men and 10 women).

Intervention(s): Eight-week lower limb resistance training period and subsequent 4-week detraining period.

Main Outcome Measure(s): Quadriceps and hamstrings concentric torque (strength), resting heart rate, arterial diameter, and blood flow velocity in the superficial femoral and carotid arteries were measured at 0, 8, 10, and 12 weeks.

Results: Resistance training increased quadriceps and hamstring strength (32% and 35%, respectively, $P < .001$), whereas strength decreased during detraining (24% and 27%, respectively, $P < .05$). Resting heart rate decreased after resistance training (16%, $P < .01$) and increased during detraining (19%, $P < .001$). Additionally, resistance training significantly increased superficial femoral and carotid resting arterial diameters (27% and 13%, respectively, $P < .001$) and mean blood flow (53% and 55%, respectively, $P < .001$). Detraining resulted in a significant decrease in superficial femoral and carotid resting diameter (46% and 10%, respectively, $P < .001$) and mean blood flow (61% and 38%, respectively, $P < .05$).

Conclusions: Resistance training initiated both local and systemic changes to arterial diameter and blood flow; these changes appeared to reverse after detraining. The local changes in response to detraining showed a worsening (beyond pretraining values) of the vascular dimensional and blood flow characteristics.

Key Words: conduit artery, resistance training, systemic circulation

Key Points

- With the appropriate duration and intensity of loading, the benefits of resistance training are comparable with the vascular adaptations to endurance exercise.
- When a contraindication to the often high-impact work associated with endurance exercise exists, resistance training may provide a suitable alternative.

Active individuals reportedly have improved vascular structure and function when compared with their sedentary counterparts.[1,2] Endurance performance is known to be limited by (among other factors) oxygen delivery and muscle perfusion, 2 aspects that can in part be modulated by an individual's vasculature.[3]

Changes in physical activity lead to acute alterations in vascular properties. An increase in physical activity, such as with endurance training, leads to increases in lumen diameter[1,2] and arterial cross-sectional area (CSA).[4] Conversely, reductions in physical activity, such as from cessation of training and bed rest, result in decreased lumen diameter and arterial CSA.[5,6] Training and detraining adaptations occur over similar timeframes[7]; however, no intermediate data over a short-duration training and detraining period are available to confirm the rate at which these changes in vascular dimensions occur. Changes in resting and exercising arterial dimensions are known to affect oxygen delivery and ultimately oxygen consumption (V_{O_2});[1,4] therefore, improvements in such dimensions could potentially enhance endurance performance. Conversely, disruptions to habitual training routines due to injury, illness, or regeneration phases could negatively affect vascular dimensions and subsequently reduce endurance performance. Resistance training offers an alternative mode of training for endurance performers experiencing such disruptions to training, and this training may in fact limit the negative effect upon vascular dimensions of a detraining period.

Although local vascular adaptations to both exercise training and physical inactivity occur, the extent of systemic vascular adaptations to training and inactivity, if any, has yet to be elucidated. Some evidence of both functional and structural systemic adaptations to exercise

training has been reported, including adaptations in the upper limbs after training emphasizing the lower body, which was attributed to elevated nitric oxide synthesis and shear stress on arterial walls.[8–11] Furthermore, evidence of systemic adaptations to inactivity is, perhaps as expected, from studies of whole-body inactivity such as bed rest,[5] whereas studies using local models of physical inactivity such as unilateral lower limb suspension (ULLS)[7] and casting[6] demonstrated solely local vascular adaptations. It is in fact suggested that the increased dependence on the nonimmobilized limbs during ULLS or lower limb casting models could prevent any expected systemic vascular adaptations, thereby producing misleading results.[12] Adopting a detraining model of deconditioning would allow for a more accurate investigation of systemic adaptations. However, to date, the only study using a detraining protocol to assess systemic adaptations involved spinal cord injury (SCI) patients.[13] Spinal cord injury patients experience progressive decreased vascularization after injury; the specific relative contribution of detraining or SCI to systemic vascular remodeling in this population remains unclear.

Despite the evident wealth of research on vascular adaptations to both exercise training and physical inactivity, the extent of local and systemic adaptations after a resistance-training protocol and subsequent detraining period in an uninjured, healthy population remains unreported. Consequently, we had 3 goals: (1) to investigate the extent of adaptations to resting arterial diameter and blood flow after a lower limb resistance-training protocol; (2) to determine the time course of any adaptations to detraining in the recently resistance-trained healthy population; and (3) to determine whether differential pattern of local versus systemic adaptations exist in both the training and subsequent detraining phases of the protocol.

During the off season, athletes experience detraining, which can lead to reduced performance and, specifically, a reduction in oxygen uptake.[4] This has been attributed to a reduction in arterial diameter, possibly due to decreased muscle blood flow.[4] Therefore, the findings from our study could provide evidence for prescribing resistance exercise during the off season to possibly assist in a more rapid return to full athletic performance on return to sport. We hypothesized that resistance training would initiate an increase in resting conduit artery diameter and blood flow, whereas detraining would reverse these increases. Additionally, we proposed that both local and systemic vasculature would be affected, with the greatest changes apparent locally.

METHODS

Study Design

This experiment was a randomized controlled trial with a mixed design, comprising a within-group measure of time (0, 8, 10, and 12 weeks), and a between-groups measure of training group (control or resistance trained). The dependent variables were isokinetic strength (maximal voluntary concentric torque about the knee joint) and vascular characteristics (blood pressure, heart rate, arterial diameter of the SFA and CA, blood flow velocity, and resistance index).

Participants

Twenty-nine healthy men and women (aged 18–38 years) volunteered to participate in the current study; 7 participants failed to adhere to the postresistance training testing protocols and were subsequently withdrawn; 1 participant withdrew for personal reasons. A total of 21 participants completed the training protocol successfully (age = 20.0 ± 2.8 years, Table 1). Volunteers were normotensive (arterial pressure range = 140/90–90/60 mm/Hg) nonsmokers and not on any medication for at least 4 weeks before the study began. Individuals currently experiencing (or having experienced during the preceding 6 months) a musculoskeletal injury were excluded from the study, as were women on all forms of hormonal contraceptives due to the potential effect of exogenous estrogen and progesterone on muscle training responses.[14] None of the participants had pursued endurance or lower limb-resistance training (as determined through a pretest questionnaire). All gave their written informed consent, and the Ethics Committee of Manchester Metropolitan University approved the study.

Experimental Procedures and Instruments

The vascular characteristics and strength of all participants were measured 4 times; before resistance training (0 weeks), after 8 weeks of resistance training (8 weeks), and after 2 and 4 weeks of detraining (10 and 12 weeks, respectively). The detraining period required all participants to terminate structured resistance training and return to their self-reported habitual physical activity levels (pretraining). The control group was instructed to maintain their habitual daily physical activities throughout the 12-week protocol and was monitored via self reports. Participants were randomly allocated to either the resistance-training (men = 5, women = 7) or control (men = 6, women = 3) group.

Resistance-Training Protocol. After we obtained baseline measurements, the experimental group performed progressive lower limb-resistance training 3 times a week over 8 weeks, totaling 24 sessions (2 gym-based and 1 home-based session each week). All 3 resistance-training sessions in the first week were supervised by an investigator, who ensured correct technique and competence for all exercises; for the remaining weeks, the investigator supervised the 2 gym-based sessions and checked diary accounts of the home-based session. Mandatory leg-training exercises included squats, Bulgarian split squats, knee extension (Pulse Fitness, Cheshire, UK), leg press (Pulse Fitness), lunges, and Samson chair (ie, static wall sit) selected based upon guidelines from previous research.[15] Before the resistance-training protocol, each participant completed a 1-repetition-maximum (1 RM) squat, lunge, leg-press, or knee-extension test under supervision; the investigator reassessed at 3 and 6 weeks to ensure the correct load was administered for each exercise. To determine 1 RM, a gradual warm-up was undertaken, and participants completed maximal lifts with a 3-minute recovery between lifts. When he or she was unable to lift the weight using the correct technique, the test was ended, and the weight before the uncompleted lift was taken as the 1 RM. Three sets of 10 repetitions of each loaded exercise were performed with a training load of 80% 1 RM. For the

Table 1. Baseline Characteristics of Training and Control Groups and Sex (Mean ± SEM)

	Group					
	Resistance Training			Control		
	Men (n = 5)	Women (n = 7)	Overall (n = 12)	Men (n = 6)	Women (n = 3)	Overall (n = 9)
Age, y	19 ± 2.2	21 ± 2.7	19 ± 3.0	21 ± 3.1	23 ± 1.1	23 ± 2.4
Systolic blood pressure, mm Hg	116 ± 2.8	113 ± 3.3	115 ± 2.2	118 ± 2.5	116 ± 1.7	117 ± 2.5
Diastolic blood pressure, mm Hg	72 ± 1.6	69 ± 1.9	70 ± 1.8	75 ± 2.2	73 ± 1.7	74 ± 1.0
Height, cm	177.3 ± 3.5	175.6 ± 6.2	176 ± 9.5	175.9 ± 3.7	171.5 ± 4.2	172.4 ± 5.9
Mass, kg	79.7 ± 3.5	74.5 ± 3.2	77.3 ± 3.9	75.8 ± 3.2	71.3 ± 3.7	73.5 ± 3.5

Samson chair exercise, 3 sets of 15 seconds using only body weight were performed. Recovery time between exercise sets was controlled at 90 seconds.

Measurements. All measurements were obtained after a 10-hour overnight fast and abstinence from caffeine and alcohol consumption. Additionally, participants did not exercise for the preceding 24 hours. Measurement of height using a manual stadiometer (model Harpenden; Holtain Ltd, Crymych, Wales, UK) and body mass using digital scales (model 91QB; DMV Electronics, Berlin, Germany) were obtained before all other measurements with participants unshod and fully clothed. Blood pressure was measured twice using an automated sphygmomanometer (model Diagnostec; Panasonic UK, Bracknell, UK) around the left upper arm after a 5-minute rest period with a minute between measures.[16] Participants were allowed to drink water ad libitum before and supplied with water on request throughout the entire testing session.

Ultrasound Measurements. After 5 minutes of prone rest in a quiet, half-darkened room to allow for regulation of vascular tone,[17] resting measurements of blood flow velocity and diameter were obtained using an echo Doppler ultrasound machine (model Technos; Esaote, Genova, Italy) with a 5.0- to 13.0-MHz broadband linear array transducer. To ensure identical probe placement during subsequent sessions, the position of the probe in relation to anatomical landmarks was traced onto a sheet of acetate. Measurement of the left SFA was obtained at 75% of femur length on the posterior thigh with the participant prone. Because this was a within-group study, we ignored limb dominance and performed all tests on the left limb, which is consistent with previous literature.[18,19] The studied limb is assumed to reflect the adaptations of both limbs. Measurement of the left CA was obtained with the participant in a seated position immediately after the SFA assessment. A perpendicular measurement from the media-adventitia interface of the near arterial wall to the lumen-intima interface of the far arterial wall was taken to determine arterial diameter. The average of 3 measurements per frame was used for artery diameter at the end-diastolic phase for both the SFA and CA. Blood flow velocity measurements were conducted using approximately 60° angle of insonation,[6,20] and the average of approximately 15 Doppler waveforms was used to calculate mean blood flow velocity. These measurements allowed for calculation of mean blood flow[7]:

$$\text{mean blood flow}\{\text{ml}\cdot\text{min}^{-1}\} = -\frac{1}{4}\pi(\text{mean diameter[cm]})^2\text{mean velocity}\left[\frac{\text{cm}}{\text{s}}\right]60\}.$$

Mean shear rate was used to estimate the more invasive measure of shear stress[21,22]:

$$\text{mean shear rate} = 4\frac{\text{mean velocity}}{\text{mean diameter}}.$$

Resting heart rate and resistance index were taken as the average from approximately 15 Doppler waveforms. Resistance index was calculated:

$$\text{resistance index} = \frac{\text{peak systolic velocity} - \text{end-diastolic velocity}}{\text{peak systolic velocity}}.$$

Measurement and analysis of all ultrasound images were conducted by the same investigator, who was blinded to the assignment of experimental and control participants using a coding system. Interday and intraday coefficients of variation (CVs) were calculated from pilot data on 4 participants. For femoral artery diameter, blood flow velocity and resistance index interday CVs were 0.25%, 1.35%, and 3.38%, respectively, and intraday CVs were 0.68%, 3.53%, and 5.04%, respectively. These values are comparable with those found in previous research and suggest high interday and intraday reliability.[1,7]

Strength Measurements. An isokinetic dynamometer (model Cybex Norm; Cybex International, Inc, Medway, MA) was used to perform maximum voluntary concentric quadriceps and hamstrings contractions (MVCs) of the right leg. The participant was seated on the dynamometer chair, with straps positioned across the shoulders and hip to avoid extraneous movements. Range of motion was processed, and the appropriate mechanical stops positioned accordingly. The dynamometer lever arm was attached to the lower limb with straps proximal to the talocrural joint, positioned at 80% of total lower leg length. The axis of rotation of the dynamometer was visually aligned to the axis of rotation of the knee joint. Full knee extension was set at 0°, and the hip was set at 90° of flexion. After a series of warm-up contractions, 5 consecutive isokinetic contractions were performed at $180°\cdot s^{-1}$, with the highest obtained result taken as the MVC.[23] Full visual (on the computer screen of the dynamometer) and verbal encouragement was provided during these efforts. This method resulted in highly reproducible isokinetic strength data with a coefficient of variation of 3.7%, which was comparable with that of previous research.[7]

Data Analysis

We conducted a repeated-measures multivariate analysis of variance to identify any strength and vascular differences between groups (control and resistance trained) and over the 12-week time period (weeks 0, 8, 10, and 12; version

18.0; SPSS Inc, Chicago, IL). The *t* test (with appropriate Bonferroni corrections) was used to perform post hoc analyses where necessary. To determine the relationship between vascular properties and isokinetic strength, we conducted Pearson correlation analyses. As an additional check on the data, the effect of sex was evaluated using the Friedman test on the raw data (with Wilcoxon post hoc tests) for within-sex analyses and Kruskal-Wallis test on the relative changes (with Mann-Whitney post hoc tests) for between-sex comparisons. Data are displayed as mean ± SEM, and statistical significance was set at $P \leq .05$.

RESULTS

Participants

A total of 21 participants completed the study. No differences existed between groups for anthropometric characteristics at baseline (Table 1) or at each measurement stage over the 12-week protocol; thus, for clarity, only baseline data are presented (Table 1). Additionally, no differences were seen between men and women; therefore, all results are reported for pooled training versus control groups.

Strength Measurement. Concentric strength of the quadriceps femoris (QF) and hamstrings changed over time for the training group ($F_{1,19} = 11.34$, $P < .001$), with mean increases of 12.03 ± 1.02 Nm from 0 to 8 weeks in QF strength ($F_{1,19} = 18.49$, $P < .001$) and 14.05 ± 0.15 Nm in hamstrings strength ($F_{1,19} = 17.09$, $P \leq .001$; Figure 1). Additionally, mean decreases in QF strength of 12.66 ± 0.95 Nm ($F_{1,19} = 4.82$, $P = .04$) and 13.87 ± 0.82 Nm in hamstrings strength ($F_{1,19} = 32.24$, $P < .001$) occurred during detraining from 8 to 12 weeks (Figure 1). No changes were observed over time in the control group ($P > .05$).

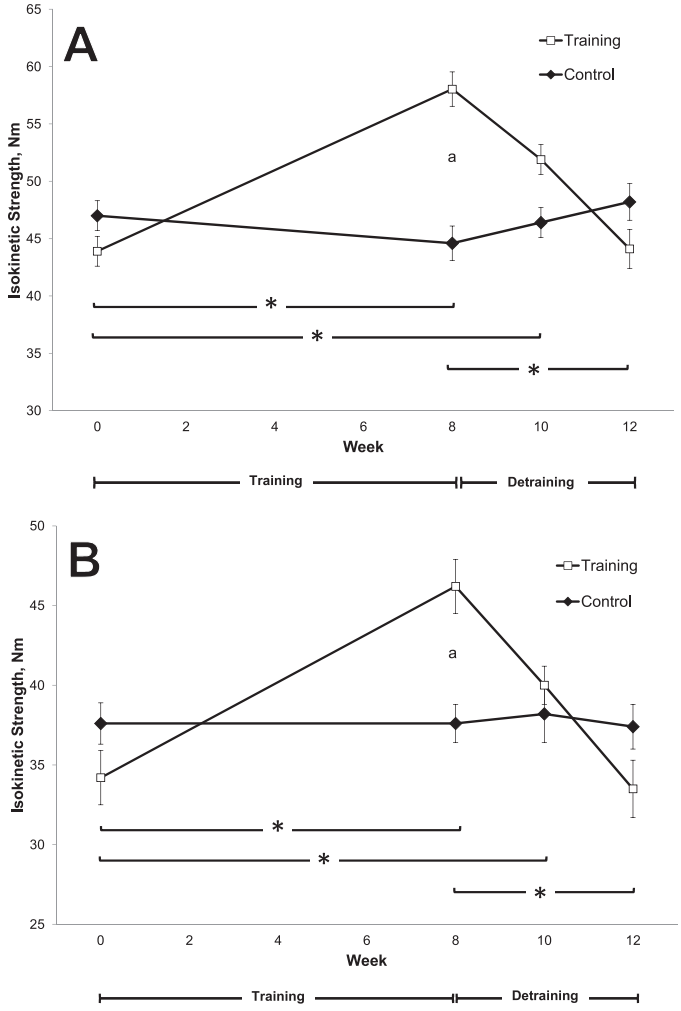

Figure 1. A, Concentric quadriceps strength and, B, hamstrings strength over 12 weeks for the training and control groups. Data are presented as mean ± SEM. *Indicates time differences, [a] group differences; $P < .05$. Concentric quadriceps and hamstring strength of the training group increased after 8 weeks of resistance training (0 to 8 weeks, $P < .001$) and decreased after 4 weeks of detraining (8 to 12 weeks, $P < .05$). No changes were observed in isokinetic strength of the control group ($P > .05$).

Heart Rate and Blood Pressure. Resting heart rate was lower at 8 weeks than at 0 weeks in the training group (16.27% ± 0.48%, $F_{1,19} = 28.55$, $P < .001$). Resting heart rate was higher than the detraining values at 10 weeks (14.5% ± 0.03%, $F_{1,19} = 13.54$, $P = .002$; Figure 2) and 12 weeks (19.64% ± 0.23%, $F_{1,19} = 32.23$, $P < .001$; Figure 2). No difference was evident between the 0- and 10-week resting heart rates in the training group ($F_{1,19} = 1.07$, $P = .31$). No changes were observed for the control group over time ($P > .05$). Furthermore, heart rate of the training group was lower than that of the control group at 8 weeks (10.8% ± 0.3%, $F_{1,19} = 7.44$, $P = .013$), whereas heart rate was greater than the control group at 10 weeks (10.20% ± 0.24%, $F_{1,19} = 7.49$, $P = .013$) and 12 weeks (13.93% ± 0.28%, $F_{1,19} = 11.62$, $P = .03$; Figure 2). Blood pressure did not differ between groups or over time ($P > .05$).

Diameter and Blood Flow of the SFA and CA. Diameters of the SFA and CA in the training group changed over time ($P < .001$ and $P \leq .005$, respectively). Mean diameter of the SFA increased 2.40 ± 0.03 mm during the training period from 0 to 8 weeks ($F_{1,19} = 52.48$, $P < .001$) and decreased 5.10 ± 0.08 mm during the detraining period from 8 to 12 weeks ($F_{1,19} = 44.39$, $P < .001$; Figure 3). Mean diameter of the CA increased 0.71 ± 0.01 mm during the training period from 0 to 8 weeks ($F_{1,19} = 33.11$, $P < .001$) and decreased 0.64 ± 0.03 mm during the detraining period between 8 to 12 weeks ($F_{1,19} = 44.5$, $P < .001$; Figure 3). No changes in SFA or CA diameter were observed for the control group over time ($P > .05$). Additionally, SFA diameter was greater for the training group than the control group at 8 weeks (2.20 ± 0.02 mm greater, $F_{1,19} = 6.20$, $P = .02$) and reduced at 12 weeks (2.90 ± 0.02 mm smaller, $F_{1,19} = 15.74$, $P \leq .001$; Figure 3). Similarly, diameter of the CA was greater for the training group than the control group at 8 weeks (0.50 ± 0.02 mm greater, $F_{1,19} = 6.63$, $P = .019$; Figure 3). Furthermore, the increase in SFA diameter during the training period between 0 and 8 weeks was greater than the increase in CA diameter ($P < .05$), and the decrease in SFA diameter during the detraining period between 8 to 12 weeks was greater than the decrease in CA diameter ($P < .05$). Both SFA and CA diameters of the training group were positively correlated with isokinetic strength over the training (8 weeks) and detraining (12 weeks) phases ($r = 0.396$, $P < .005$ and $r = 0.597$, $P < .05$, respectively).

Mean resting blood flow of the training group for the SFA and CA increased between 0 and 8 weeks (by 180.7 ± 12.2

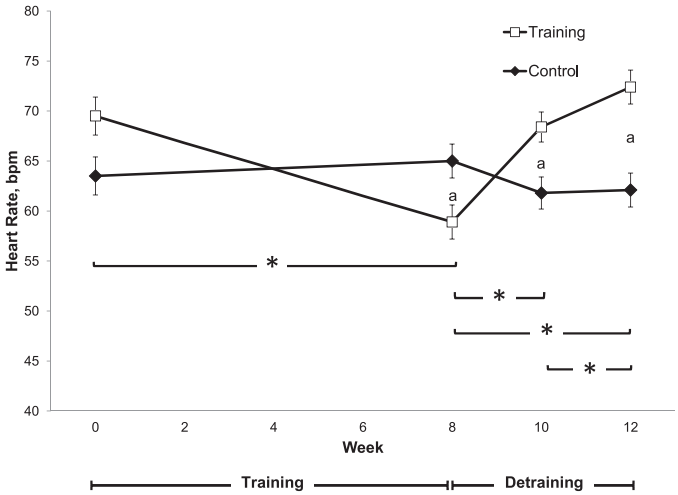

Figure 2. Resting mean heart rate over 12 weeks for training and control groups. Data are presented as mean ± SEM. *Indicates time differences, [a] group differences; $P < .05$. Resting heart rate of the training group decreased after 8 weeks of resistance training (0 to 8 weeks, $P < .001$) and increased after 4 weeks of detraining (8 to 12 weeks, $P < .001$). No changes in resting heart rate of the control group were observed over time ($P > .05$).

ml·min^{-1}, $F_{1,19} = 23.99$, $P < .001$and 221.0 ± 26.5 ml·min^{-1}, $F_{1,19} = 16.56$, $P < .001$, respectively; Figure 3). Mean resting blood flow of the training group decreased by 200.0 ± 11.1 ml·min^{-1} during the detraining period between 8 and 10 weeks for the SFA ($F_{1,19} = 9.93$, $P \leq .005$) and by 234.0 ± 35.2 ml·min^{-1} for the CA ($F_{1,19} = 7.41$, $P \leq .005$). Subsequently, a decrease in resting mean blood flow of 28.0 ± 1.5 ml·min^{-1} between 10 and 12 weeks occurred for the SFA ($F_{1,19} = 4.75$, $P = .042$), whereas an increase of 50.8 ± 6.6 ml·min^{-1} was observed for the CA ($F_{1,19} = 5.61$, $P = .029$; Figure 3). No changes in SFA or CA resting blood flow were observed for the control group over time ($P > .05$). Group differences in postexercise mean blood flow of 154.2 ± 4.701 and 205.2 ± 4.6 ml·min^{-1} existed at 8 weeks for the SFA ($F_{1,19} = 5.74$, $P = .027$) and CA ($F_{1,19} = 7.41$, $P = .014$), respectively, and of 94.0 ± 4.9 ml·min^{-1} at 12 weeks for the CA only ($F_{1,19} = 5.62$, $P = .029$; Figure 3). No differences in blood flow between SFA and CA at 8 weeks were observed ($P > .05$); however, blood flow in the CA decreased more than that in the SFA at 12 weeks ($P < .05$). Superficial femoral artery diameter was positively correlated with SFA mean blood flow ($r = 0.838$, $P < .001$) and CA diameter was positively correlated with CA mean blood flow ($r = 0.385$, $P < .001$). Mean shear rate of the training

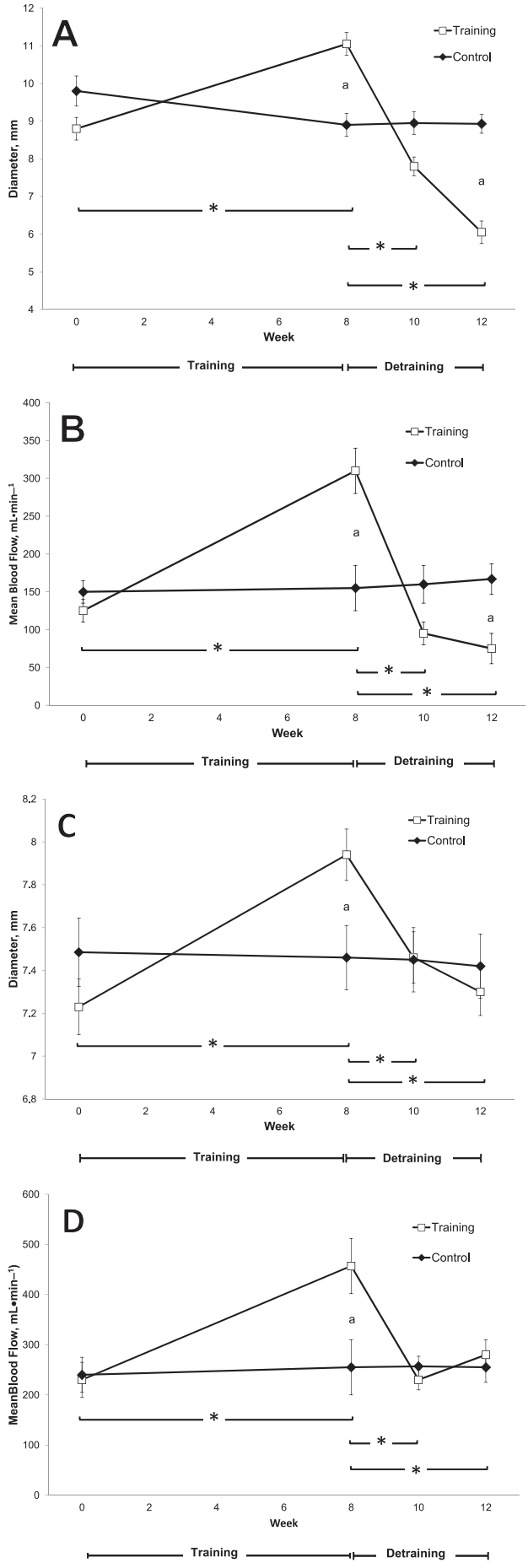

Figure 3. Resting mean diameter of the A, superficial femoral artery (SFA), and B, carotid artery (CA) over 12 weeks for the training and control groups. Resting mean blood flow for the B, SFA, and D, CA over 12 weeks for the training and control groups. Data are presented as mean ± SEM. *Indicates time differences, [a] group differences; $P < .05$. Diameter of the SFA and CA increased after 8 weeks of resistance training between 0 and 8 weeks ($P < .001$) and decreased after detraining between 8 and 12 weeks ($P < .001$). Mean blood flow of the SFA and CA increased from 0 to 8 weeks ($P < .001$). After 2 weeks of detraining, mean blood flow of the SFA and CA decreased ($P \leq .005$). However, mean blood flow of the SFA decreased further from 10 to 12 weeks ($P < .05$), whereas mean blood flow of the CA increased during this time ($P < .05$).

group for the SFA and CA increased between 0 and 8 weeks (by 13.25 ± 0.4 s^{-1}, $F_{1,19} = 4.35$, $P = .036$ and 21.56 ± 1.3 s^{-1}, $F_{1,19} = 7.84$, $P = .042$, respectively; Figure 4). Both SFA and CA mean shear rates correlated with isokinetic strength over the training and detraining phases ($r = 0.489$, $P < .005$ and $r = 0.584$, $P < .005$). Mean resistance index of the SFA for the training group decreased by 0.02 during the training period between 0 and 8 weeks ($F_{1,19} = 18.46$, $P < .001$) and increased by 0.03 during the detraining period between 8 and 12 weeks ($F_{1,19} = 3.64$, $P = .001$; Figure 4). The resistance index did not differ between groups for the CA at rest or for the control group over time ($P > .05$). Group differences in mean SFA resistance index were evident at 8 weeks (0.01, $P < .05$), 10 weeks (0.01, $P < .05$), and 12 weeks (0.02, $P < .05$; Figure 5). A decrease in the resistance index of the CA at rest for the training group occurred between 0 and 10 weeks (0.01, $P < .05$).

Effect of Sex. Differences in strength and vascular factors (diameter, blood flow, estimated shear rate, and resistance index) were observed over time within both the men and women ($X^2_{39} = 74.1$, $P < .001$ and $X^2_{39} = 81.5$, $P < .001$, respectively; Table 2a). No differences were noted over time for either men or women in the control group ($P > .05$; Table 2b). Differences were seen between men and women in the training group in the baseline-normalized change in estimated shear rate for the SFA and CA only at 10 weeks ($P < .05$). Similarly, differences were evident between men and women for the baseline-normalized change in resistance index at 10 and 12 weeks ($P < .05$). No other sex differences in the baseline-normalized changes (including strength and the remaining vascular factors) were observed in either the training ($P > .05$, Table 3a) or the control group ($P > .05$, Table 3b).

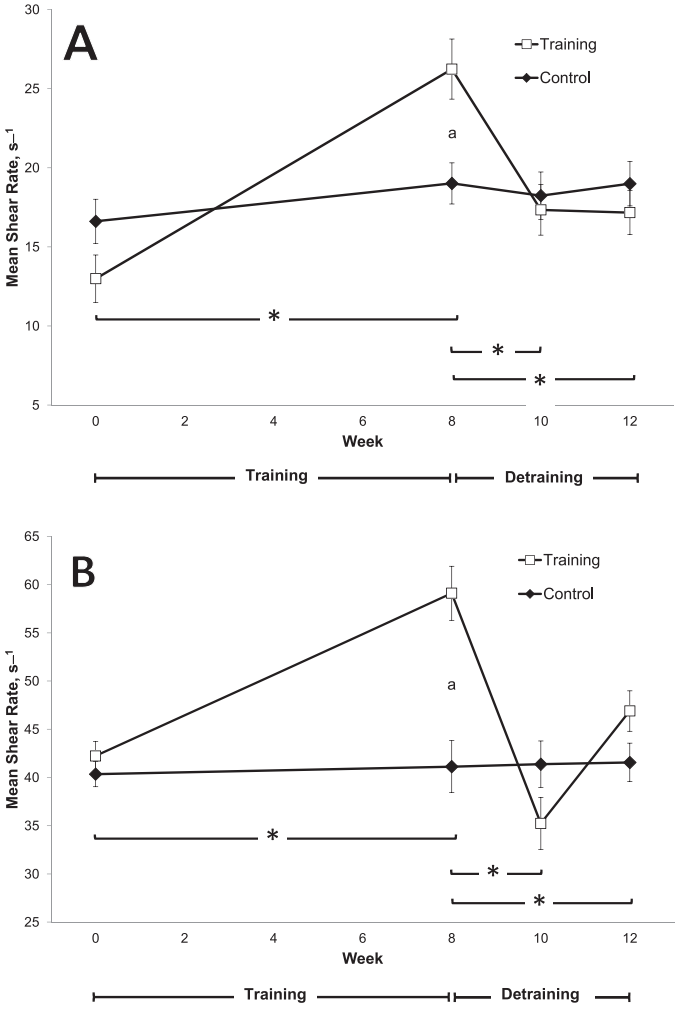

Figure 4. Estimated mean shear rate for the, A, superficial femoral artery and, B, carotid artery over 12 weeks for the training and control groups. Data are presented as mean ± SEM. *Indicates time differences, [a] group differences; $P < .05$. Mean shear rate of the superficial femoral artery and carotid artery increased ($P < .05$) from 0 to 8 weeks for the training group and decreased ($P < .05$) after 4 weeks of detraining from 8 to 12 weeks.

DISCUSSION

We investigated the extent and time course of adaptations in resting arterial diameter and blood flow to resistance training and detraining in a young, healthy population. We hypothesized that resistance training would augment resting arterial diameter and mean blood flow, whereas detraining would reverse these. Additionally, we proposed that both local (ie, SFA) and systemic (ie, CA) changes to training and detraining would occur, with the greatest changes expected locally. Our findings confirmed these hypotheses: changes occurred in local and systemic arterial structure and flow during the training and detraining periods.

Effectiveness of Resistance Training and Detraining

The training program adopted for this study was sufficient to induce lower limb conditioning, demonstrated by increases in QF and hamstrings strength (32% and 35%, respectively). These increases are similar in magnitude to those reported previously for similar-duration training studies (30% and 25% for QF and hamstrings, respectively).[24,25] Furthermore, we noted decreases in QF (24%) and hamstrings (27%) strength during the detraining period, which suggests the detraining period was sufficient to show

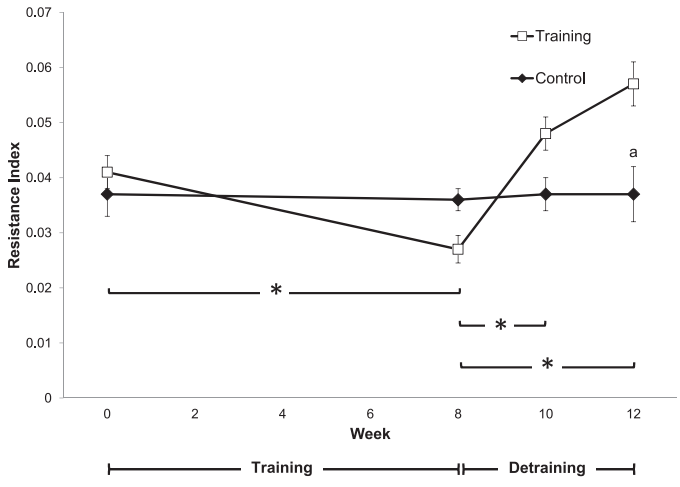

Figure 5. Mean resistance index for the superficial femoral artery over 12 weeks for the training and control groups. Data are presented as mean ± SEM. *Indicates time differences, [a] group differences; $P < .05$. Mean resistance index of the training group decreased after 8 weeks of resistance training (0 to 8 weeks, $P < .001$) and increased after 4 weeks of detraining (8 to 12 weeks, $P \leq .001$). No changes in mean resistance index of the control group were observed over time ($P > .05$).

Table 2a. Strength and Vascular Changes in the Resistance-Training Group Over Time by Sex (Mean ± SEM)

	Time, wk							
	0		8		10		12	
Variable	Men	Women	Men	Women	Men	Women	Men	Women
Isokinetic strength, Nm								
Quadriceps	45.35 ± 3.51[ab]	42.82 ± 1.98[ab]	61.29 ± 3.67[bc]	57.02 ± 2.17[bc]	53.73 ± 2.45[ac]	49.48 ± 1.93[ac]	45.21 ± 3.62[ab]	43.40 ± 1.94[ab]
Hamstrings	34.83 ± 3.27[ab]	33.67 ± 2.15[ab]	47.54 ± 3.077[bc]	45.92 ± 1.90[bc]	40.51 ± 2.39[ac]	39.67 ± 1.98[ac]	34.48 ± 4.14[ab]	32.85 ± 2.04[ab]
Resting heart rate, b·min^{-1}	69.32 ± 2.11[a]	70.26 ± 2.73[a]	59.44 ± 1.92[bc]	58.07 ± 1.68[bc]	69.52 ± 1.16[a]	67.79 ± 1.59[a]	70.54 ± 1.28[a]	73.2 ± 2.32[a]
Diameter, mm								
SFA	8.84 ± 0.04[ab]	8.76 ± 0.01[ab]	11.06 ± 0.03[bc]	11.04 ± 0.02[bc]	7.80 ± 0.02[ac]	7.70 ± 0.03[ac]	6.03 ± 0.01[abc]	6.06 ± 0.01[abc]
CA	7.24 ± 0.01[ab]	7.22 ± 0.01[ab]	7.96 ± 0.01[bc]	7.94 ± 0.01[bc]	7.48 ± 0.01[ac]	7.43 ± 0.01[ac]	7.32 ± 0.01[abc]	7.27 ± 0.01[abc]
Blood flow, mL·min^{-1}								
SFA	126.24 ± 2.25[ab]	123.95 ± 1.08[ab]	311.15 ± 1.40[bc]	308.76 ± 1.10[bc]	96.14 ± 1.12[ac]	93.95 ± 1.03[ac]	77.25 ± 1.58[abc]	74.37 ± 1.19[abc]
CA	232.36 ± 1.89[ab]	229.84 ± 1.51[ab]	459.47 ± 3.18[bc]	455.95 ± 1.39[bc]	230.74 ± 1.60[a]	228.49 ± 1.98[a]	282.03 ± 2.05[abc]	279.17 ± 1.38[abc]
Estimated mean shear rate, s^{-1}								
SFA	13.76 ± 0.41[ab]	12.54 ± 0.17[ab]	27.30 ± 0.28[bc]	26.02 ± 0.14[bc]	16.94 ± 0.21[ac]	17.64 ± 0.22[ac]	17.03 ± 0.23[abc]	17.84 ± 0.20[abc]
CA	44.45 ± 0.44[ab]	41.74 ± 0.28[ab]	60.26 ± 0.52[bc]	58.01 ± 0.36[bc]	34.69 ± 0.63[ac]	35.90 ± 0.20[ac]	47.56 ± 0.24[abc]	44.72 ± 0.25[abc]
Resistance index	0.042 ± 0.00[ab]	0.039 ± 0.00[ab]	0.027 ± 0.00[c]	0.027 ± 0.00[c]	0.047 ± 0.00[c]	0.048 ± 0.00[c]	0.058 ± 0.00[abc]	0.057 ± 0.00[abc]

Abbreviations: CA, carotid artery; SFA, superficial femoral artery.
[a] Different from 0 weeks ($P \leq .05$).
[b] Different from 8 weeks ($P \leq .05$).
[c] Different from 10 weeks ($P \leq .05$).

Table 2b. Strength and Vascular Changes in the Control Group Over Time by Sex (Mean ± SEM)

	Time, wk							
	0		8		10		12	
Variable	Men	Women	Men	Women	Men	Women	Men	Men
Isokinetic strength, Nm								
Quadriceps	48.62 ± 3.41	46.14 ± 3.12	45.08 ± 3.35	44.25 ± 2.73	46.24 ± 2.84	45.72 ± 3.11	48.91 ± 3.63	46.56 ± 2.54
Hamstrings	38.1 ± 2.84	36.23 ± 1.70	38.34 ± 2.45	37.15 ± 1.92	38.93 ± 3.18	37.71 ± 2.24	38.02 ± 2.77	36.96 ± 2.03
Resting heart rate, b·min^{-1}	64.02 ± 2.41	63.11 ± 1.62	65.72 ± 1.86	64.33 ± 1.31	62.68 ± 2.05	61.13 ± 1.64	63.34 ± 2.34	61.61 ± 1.98
Diameter, mm								
SFA	9.96 ± 0.02	9.72 ± 0.01	9.02 ± 0.02	8.70 ± 0.02	8.93 ± 0.03	9.04 ± 0.01	8.92 ± 0.02	8.97 ± 0.02
CA	7.52 ± 0.01	7.39 ± 0.01	7.48 ± 0.02	7.44 ± 0.01	7.45 ± 0.01	7.44 ± 0.01	7.42 ± 0.01	7.43 ± 0.01
Blood flow, mL·min^{-1}								
SFA	154.74 ± 2.25	149.9 ± 1.52	157.14 ± 2.41	153.59 ± 1.73	160.48 ± 1.96	157.25 ± 1.58	162.82 ± 2.11	160.97 ± 1.92
CA	242.86 ± 1.46	239.16 ± 1.67	255.16 ± 0.92	253.28 ± 1.93	258.83 ± 1.24	255.07 ± 1.68	257.70 ± 1.75	250.30 ± 1.25
Estimated mean shear rate, s^{-1}								
SFA	17.35 ± 0.32	16.52 ± 0.27	19.21 ± 0.47	18.25 ± 0.28	18.83 ± 0.37	17.56 ± 0.31	19.26 ± 0.25	18.11 ± 0.34
CA	40.73 ± 0.45	39.25 ± 0.36	41.82 ± 0.32	40.26 ± 0.26	41.95 ± 0.33	40.67 ± 0.21	42.13 ± 0.27	41.09 ± 0.23
Resistance index	0.037 ± 0.00	0.036 ± 0.00	0.036 ± 0.00	0.036 ± 0.00	0.037 ± 0.00	0.036 ± 0.00	0.037 ± 0.00	0.037 ± 0.00

Abbreviations: CA, carotid artery; SFA, superficial femoral artery.

Table 3a. Between-Sexes Comparison of the Magnitude of Strength and Vascular Changes (Relative to Baseline [0 weeks]) in the Resistance-Training Group (n = 12), % (Mean ± SEM)

	Time, wk					
	8		10		12	
Variable	Men	Women	Men	Women	Men	Women
Isokinetic strength, Nm						
Quadriceps	35.15 ± 2.71	33.16 ± 4.71	18.48 ± 2.26	15.55 ± 2.33	−0.31 ± 0.51	1.35 ± 0.49
Hamstrings	36.49 ± 2.40	36.38 ± 2.56	16.31 ± 2.55	17.82 ± 2.75	−1.00 ± 0.98	−2.44 ± 0.60
Resting heart rate, b·min^{-1}	−14.25 ± 0.73	−17.35 ± 0.90	0.29 ± 1.18	−3.52 ± 1.23	−1.76 ± 0.74	4.18 ± 0.76
Diameter, mm						
SFA	25.11 ± 0.13	26.03 ± 0.10	−11.76 ± 0.18	−12.10 ± 0.20	−31.79 ± 0.11	−30.82 ± 0.07
CA	9.94 ± 0.05	9.97 ± 0.04	3.31 ± 0.09	2.91 ± 0.10	1.10 ± 0.09	0.69 ± 0.08
Blood flow, mL·min^{-1}						
SFA	146.47 ± 1.31	149.10 ± 0.94	−23.84 ± 0.44	−24.20 ± 0.47	−38.81 ± 0.34	−40.00 ± 0.31
CA	97.74 ± 0.50	98.38 ± 0.52	−0.70 ± 0.28	−0.59 ± 0.30	21.38 ± 0.22	21.46 ± 0.15
Estimated mean shear rate, s^{-1}						
SFA	46.61 ± 0.41	50.73 ± 0.29	23.66 ± 1.91[a]	37.99 ± 1.10[a]	22.92 ± 1.67	34.19 ± 1.12
CA	36.38 ± 0.74	35.13 ± 0.98	−21.54 ± 0.68[a]	−17.54 ± 0.61[a]	7.32 ± 0.50	7.03 ± 0.63
Resistance index	−32.50 ± 0.68	30.77 ± 0.36	17.50 ± 1.42[a]	23.08 ± 0.45[a]	45.00 ± 1.45[a]	46.15 ± 1.24[a]

Abbreviations: CA, carotid artery; SFA, superficial femoral artery.
[a] Indicates $P \leq .05$.

Table 3b. Between-Sexes Comparison of the Magnitude of Strength and Vascular Changes (Relative to Baseline [0 weeks]) in the Control Group (n = 9), % (Mean ± SEM)

	Time, wk					
	8		10		12	
Variable	Men	Women	Men	Women	Men	Women
Isokinetic strength, Nm						
Quadriceps	−7.28 ± 2.24	−4.10 ± 3.71	−4.90 ± 3.84	−0.91 ± 3.12[a]	−0.60 ± 2.52	0.91 ± 2.83
Hamstrings	0.63 ± 1.25	2.54 ± 1.84	2.18 ± 1.3	4.09 ± 2.02	−0.21 ± 1.11	2.01 ± 1.76
Resting heart rate, b·min^{-1}	2.66 ± 0.82	1.93 ± 0.68	−2.09 ± 1.14	−3.14 ± 1.06	−1.06 ± 0.83	−2.38 ± 1.22
Diameter, mm						
SFA	−9.44 ± 0.10	−10.49 ± 0.08	−10.34 ± 0.13	−7.00 ± 0.18	−10.44 ± 0.09	−7.72 ± 0.08
CA	−0.53 ± 0.07	0.68 ± 0.06	−0.93 ± 0.08	0.68 ± 0.08	−1.33 ± 0.11	0.54 ± 0.07
Blood flow, mL·min^{-1}						
SFA	1.55 ± 1.48	2.46 ± 1.03	3.71 ± 0.78	4.90 ± 0.85	5.22 ± 0.42	7.38 ± 0.53
CA	5.39 ± 0.63	5.90 ± 0.44	6.58 ± 0.37	6.65 ± 0.38	6.11 ± 0.31	4.66 ± 0.34
Estimated mean shear rate, s^{-1}						
SFA	10.72 ± 0.53	10.47 ± 0.56	8.53 ± 1.54	6.30 ± 1.49	11.01 ± 1.36	9.62 ± 1.21
CA	2.68 ± 0.86	2.57 ± 0.92	3.00 ± 1.12	3.62 ± 0.74	3.44 ± 0.65	4.69 ± 0.89
Resistance index	−2.70 ± 0.45	0.00 ± 0.67	0.00 ± 1.32	0.00 ± 0.78	0.00 ± 1.12	2.78 ± 1.42

Abbreviations: CA, carotid artery; F, female; M, male; SFA, superficial femoral artery.
[a] Indicates $P \leq .05$.

lower limb deconditioning. Similarly, Schulze et al[26] reported a 22% decrease in isokinetic quadriceps MVC after 3 weeks of ULLS.

Resistance Training and Arterial Measurements. In our investigation, 8-week resistance training caused an increase in diameter of the SFA diameter (27%) compared with the CA (13%). Unfortunately, data on resistance-training adaptations to the SFA and CA remain limited. However, similar increases in local arterial diameter have been observed in studies using aerobic exercise.[1,4] The occurrence of both local and systemic changes in arterial diameter and blood flow in our investigation appears comparable with previous reports of systemic adaptations in upper limb vessels after lower limb training.[9] However, systemic adaptations to local conditioning have only been observed in studies using a combination of aerobic and resistance training.[9]

The stimuli for the local and systemic increases in arterial diameter and blood flow we observed may be attributed to changes in endothelial dilatory factors or systemic rises in pressure. In response to exercise training or physical inactivity, functional vascular adaptations occur initially but are soon superseded by structural adaptations to the vessel walls,[27] with arterial remodeling thought to be dependent on both shear stress and nitric oxide (NO).[28] During acute exercise training, endothelial NO synthase (eNOS) expression and subsequent NO production increase to moderate the elevated shear stress on the endothelial surface.[29] With prolonged exercise training, the enhanced NO levels are believed to initiate structural vessel remodeling to adjust shear levels back to a new normal level at which they are maintained.[29] We cannot determine whether structural changes occurred, because the increased diameter could indeed be attributed to acute changes in NO sensitivity as outlined by Tuttle et al,[27] rather than a remodeling process of the arterial wall. However, in short-term (12-week) training studies, remodeling of the arterial intima-media thickness has been observed,[1] which suggests that the increase in arterial diameter may have occurred from a combination of changes in endothelial dilatory factors or structural remodeling. In addition to changes in dilatory factors and arterial structure, a further contributory factor to increased arterial diameter could be an increase in blood volume. Blood volume increases with training and contributes to increases in blood flow and cardiac output observed with training. Hydration levels cause acute changes in vessel diameter: clinical dehydration (defined as a $\geq$5% reduction in body weight) reduced vessel diameter by 2.7 mm.[30] We did not measure the dehydration status of our participants before testing sessions; however, all of our participants were healthy individuals and were allowed to drink water ad libitum before and during testing. Hence, we assumed that they were not dehydrated and that hydration status did not differ among testing sessions. Indeed, large variations in hydration status are typically seen only within clinical populations. Additionally, all participants demonstrated an increase in SFA diameter with training (mean = 2.40 $\pm$ 0.03 mm) and a decrease with detraining (mean = 5.10 $\pm$ 0.08 mm), both of which were beyond what would be expected for any day-to-day variations in hydration status.[30]

Shear stress on the endothelial cells is caused by fluctuations in blood flow associated with muscle hypertrophy.[22] The moderate association between conduit arterial diameter and strength in the current study may be explained by muscle hypertrophy. We did not measure muscle mass, although similar resistance-training protocols have resulted in hypertrophy.[31] In fact, previous authors[22] proposed that the increased blood flow associated with hypertrophy could be explained by enhanced capillary or arteriolar proliferation (or both). We noted a significant correlation between arterial diameter and blood flow, which suggests that changes in shear stress were responsible for the observed changes in arterial diameter. We recognize that arterial diameter is directly proportional to blood flow, so we propose a causal relationship whereby the incremented blood flow instigates increased shear stress, itself initiating vessel adaption through increased diameter. Additionally, pressure and circumferential wall stress could be other primary mechanisms involved in vessel remodeling.[32] Although our methods did not allow for direct measurement of shear stress, pressure, or circumferential stress, it is likely that all of these mechanisms are involved in the observed changes in arterial diameter. Despite this limitation, our estimation of shear rate (an estimate of shear stress), changed over time and between groups. Furthermore, shear rate correlated with strength, thus suggesting that an exercise-induced increase in shear stress may indeed be 1 mechanism involved in the observed changes in arterial diameter. However, calculation of shear rate is based upon the Poiseuille law, which assumes that a Newtonian fluid is flowing through straight, stiff arteries, even though this is not entirely true of arterial circulation.[21]

Moreover, the extent of shear stress experienced with exercise is influenced by anterograde and retrograde oscillatory blood flow.[32] Anterograde blood flow produces greater shear stress than does retrograde blood flow.[32] The effects of exercise in vivo are complex, reflecting the nonuniformity of the arterial tree combined with the effect of muscle contractions. Resistance exercise reduces retrograde blood flow in the arteries perfusing active muscle beds and increases retrograde flow in the nonexercising or inactive arteries.[32] Hence, this may explain the observed increased diameter of the SFA in our study but not the CA, which may be attributed to increased pressure and circumferential stress. Acute bouts of resistance training elevate blood pressure, which causes circumferential stress in compliant arteries as the endothelial cells stretch to compensate.[33] When intraluminal pressures exceed 135 mm Hg, eNOS expression increases and NO content is elevated.[32] Although we did not measure intraluminal pressure during the exercise bouts, Fleck et al[33] demonstrated that novice resistance trainers experienced blood pressures between 130 and 200 mm Hg during lower limb resistance exercises at 80% 1 RM, which is the same intensity we used. This suggests that our participants experienced exercising blood pressures within this range, which may have subsequently elevated eNOS expression and NO synthesis, resulting in arterial remodeling. Furthermore, a concomitant decrease in resting heart rate of 16% was observed during the training period, followed by an increase of 19% during the detraining period. This may be explained by changes in cardiac output subsequent to alterations in conduit arterial diameter and a reduction in peripheral resistance, changes that are similar in magnitude to previous reports.[33]

Detraining and Arterial Measurements. Seemingly, during detraining or periods of physical inactivity, inward arterial remodeling occurs, resulting in augmented shear levels.[5,7] In our study, the effects of detraining were apparent both locally and systemically, with a 46% decrease in SFA diameter and 11% decrease in CA diameter apparent from 8 to 12 weeks. Most of these changes (30% and 7% for SFA and CA, respectively) occurred during the initial 2 weeks of the detraining period. This finding is comparable with previous research and suggests the majority of vascular remodeling occurs rapidly after exercise training ends.[6]

Most authors studying deconditioning have measured vascular adaptations from baseline, whereas we investigated the effect of deconditioning in a recently trained population. Unsurprisingly, the changes in arterial diameter we observed are greater than those in previous reports.[5,7] These differences may be explained by a greater responsiveness to arterial remodeling in the trained population, probably due to an upregulation of eNOS and subsequent NO synthesis compared with sedentary participants and may also explain why SFA diameter decreased below baseline values at 12 weeks in the training group.[34] Furthermore, all of the aforementioned authors recruited either sedentary or symptomatic populations to investigate the extent of deconditioning on the vasculature. Understanding the extent of vascular adaptations to deconditioning in recently trained individuals has implications for athletic populations experiencing injury or illness and may be useful to the development of exercise countermeasures during such deconditioning periods.

Local Versus Systemic Changes. We found that the local changes in arterial diameter were greater than the systemic changes as a result of both the training and detraining periods. This result may be attributed to greater eNOS expression after resistance training and subsequent elevated local NO levels.[4] Additionally, Thijssen et al[12] suggested that conduit arteries supplying appendicular muscle groups demonstrate greater adaptability than conduit arteries supplying axial regions. Taken together, these findings may explain the greater changes in arterial diameter observed locally than systemically.

To date and to our knowledge, we are the first to report systemic changes in arterial diameter and blood flow after a local deconditioning protocol. Previous authors[6,7] using local deconditioning methods have failed to report systemic arterial remodeling, possibly because they indirectly increased reliance on the nonimmobilized upper limbs during ambulation, which could prevent any systemic deconditioning effects. Furthermore, the systemic changes in arterial diameter in our study were almost completely reversed during the detraining period and arterial diameter did not decrease below baseline, which is in agreement with previous reports.[22] Future researchers may extend the detraining period to determine if further changes to arterial diameter and blood flow occur with deconditioning or if the systemic vasculature is sensitive only to adaptations above baseline.

Effect of Body Mass and Sex on Observations. As previously reported, arterial diameter is determined by age, sex, and body mass.[35] However, vascular adaptations to training without changes to body mass would suggest a local adaptation to blood flow and vessel properties resulting from a training intervention.[5,36] Our data confirm these observations and suggest that changes in vessel diameter and blood flow can occur without any modification to body mass. Although sex differences at baseline have previously been reported in blood flow and vessel diameter,[35,37] we observed no difference between sexes in the absence of training. Similarly, the changes with training and detraining suggested a similarity in the adaptations exhibited by both sexes (after training SFA diameter: men = 25% versus women = 26%, $P > .05$; CA diameter: men = 9% versus women = 9%, $P > .05$; after detraining SFA diameter in men = –32% versus women = –31%, $P > .05$; CA diameter in men = 1% versus women = 1%, $P > .05$). Previous reports[37] of sex differences in vessel diameter and blood flow have been attributed to differences in body mass, but this was not the case in our study population because the men and women had similar body mass. It is, however, debatable whether to expect a sex difference in vascular responses as a result of resistance training or detraining (or both) in humans; the only report[38] of sex differences to date has been observed in porcine arteries. Our investigation was not powered to identify a sex difference; therefore, to follow up on this specific issue of a potential effect of sex on findings would be timely.

CONCLUSIONS

Resting diameter and blood flow of the SFA and CA increased after lower limb resistance training, possibly indicating that this mode of training initiates both local and systemic conduit artery remodeling. This would subsequently improve athletic performance through potential for better oxygen delivery and muscle perfusion.[1,4] After detraining, a reversal of these training changes occurred, with reductions to below baseline levels apparent in SFA diameter and CA blood flow. Most of these changes occurred within the initial 2 weeks of the detraining period, suggesting that both resting arterial diameter and blood flow changes occur rapidly once exercise training ends. It is therefore evident that exercise and inactivity affect the human vasculature; however, these adaptations do not seem to occur linearly. Thus, exercise should be prescribed to trained individuals experiencing detraining periods as dictated by a number of factors, including the off season, a short-term injury, or a debilitating illness. Preventing or slowing these detraining adaptations will decrease the time required to return to full athletic performance. Future researchers should study an exercise countermeasure to minimize the adverse vascular adaptations that occur during a detraining period.

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